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UNITED STATES.

[Reports to the Surgeon-General United States Marine-Hospital Service.]

THE EPIDEMIC DYSENTERY OF THE PAST TWENTY YEARS IN JAPAN.

SIR: In accordance with your instructions, I have the honor to submit the following report on the epidemic dysentery now prevalent and, for twenty years past, annually recurring in this country:

The records of epidemic dysentery in Japan begin in 1878, about which time the Government first attempted to collect statistics of disease; and I am unable to find an account of any serious epidemic, which may reasonably be supposed to have been of dysentery, at any earlier period.

Up to 1880 the disease prevailed only in the southern island of Kiushiu, in the kens of Nagasaki, Oita, Kumamoto, and Fukuoka (see map No. 1). In 1881 it invaded the whole island of Shikoku, with its four prefectures of Yehime, Kochi, Kagawa, and Tokushima. In 1882, crossing the western strait, it attacked somewhat severely the southern ken of the island of Nippon, that of Yamaguchi, whence it gradually advanced to the northeast, and later northward, rather irregularly and occasionally by leaps, though generally moving in the lines of most active intercommunication, until, in 1897, every province of the three large islands of Nippon, Shikoku, and Kiushiu was included in the area of the epidemic.

The territorial expansion of the field of the disease was, however, checked for a brief time, beginning with 1886, at a mountain chain trending from east to west, and lying just south of the ancient capital, Kioto.

In 1890–91 the advance of the disease began again, continuing until, in 1896–97, it was temporarily arrested by the Strait of Tsugar, between Nippon and the northern island of Yezo, or the Hokkaido. This barrier has been passed during the present season of 1899, and there is every prospect of a more or less permanent invasion of the Hokkaido.

For convenience sake, I give herewith a sketch map of Japan (map No. 1), showing the present division of the Empire into fu or municipal jurisdictions, but 3 in number and surrounding and including the great cities of Kioto, Osaka, and Tokyo; and ken, which correspond closely to the prefectures of France. These do not appear on any map of Japan yet printed for the use of foreigners. The date of serious invasion by dysentery is given on the map for each of these geographical divisions, and the, on the whole, steady and persistent progress of the epidemic is thus shown at a glance.

The statistics of the disease from 1878 to November 10, 1899, are as follows (see also chart).

Year.	Cases.	Deaths.	Year.	Cases.	Deaths.	Year.	Cases.	Deaths.
1878	1,118	206	1886	24,326	6,839	1894	155,140	38,094
1879	8,322	1,487	1887	16,149	4,257	1895	52,711	12,959
1880	5,047	1,305	1888	26,815	6,576	1896	85,876	22,356
1881	7,001	1,837	1889	22,873	5,970	1897	91,077	23,189
1882	4,330	1,313	1890	42,633	8,706	1898	90,933	22,379
1883	21,172	5,066	1891	46,358	11,208	1899	125,989	26,709
1884	22,702	6,036	1892	70,842	16,844			
1885	47,377	10,690	1893	167,305	41,282			

A total of 1,136,096 cases and of 275,308 deaths, the percentage of mortality for the whole period being 24.23.

It is probable that, up to 1883, the epidemic in the districts then affected was somewhat more severe than as shown in the foregoing table, for the system of reports was not yet fully protected; even now it is certain that the statistics of the Government include but the larger proportion of the cases. In rural districts, which are specially affected by the disease, not only do many cases pass unreported, but deaths by it are likely, through ignorance or desire for concealment, to be ascribed to other causes, and, everywhere, many of the slighter cases escape official notice.

Even without making allowance for understatement, the statistics are sufficiently appalling. An epidemic of annual recurrence, which has since 1890 averaged a morbidity of 92,854 and a mortality of 22,345, or 24.06 per cent, is certainly serious enough to demand every effort on the part of state medicine for its control.

The rate of mortality throughout the whole of the prolonged epidemic has been remarkably constant, ranging between 24 and 27 per cent. The latter higher figure marked the earlier period of the outbreak, 1878–1882, but the real proportional mortality of these years was probably somewhat less, as at that time it was the graver cases only which were officially returned. A study of the statistics of more recent years, not only of the country at large, but those of the provinces one by one, has convinced me that the regular and, so to speak, normal, mortality of the disease as manifested here is close to 25 per cent.

An idea of the proportion in which it has affected the population during the past few years can be gathered from the accompanying map (No. 2), showing the number attacked in 10,000 of the people, in each fu and ken, during the epidemic of 1898, when the central provinces suffered most severely, the small ken of Yamanashi alone having over 10,000.

In the earlier years of the prevalence of the epidemic, neither the Government nor the people realized its present and growing importance, and although a very good code of regulations for the prevention of infectious disease was promulgated in 1880, it was not until 1885 that the authorities seemed to take up the question of the controlling of dysentery, for the first time, with any energy.

At this time, and for some years later, sanitary administration was entrusted to the hands of the local governments, a policy which proved disastrous, not only as regards the management of dysentery, but repeatedly appeared to be responsible for the admission of cholera from abroad. In 1895 the central government assumed control of all matters connected with state medicine, but difficulties as to appropriations and awkward division of responsibility between the officers of the general government and local authorities, greatly interfered with the work of the sanitary bureau of the home department. A somewhat similar state of affairs is not unknown in the United States.

In 1897 the Regulations for the Prevention of Infectious Disease were revised, and the central government has now direct and complete authority, hampered only by deficiency of funds. Want of money, in many instances, compels the employment of incompetent men in positions requiring trained and experienced sanitarians. Nevertheless, remarkably plucky and skillful work is accomplished even by the ordinary police force, a body which, in Japan, fills many functions.

So far but little success has attended the efforts of the officials to control the disease or to limit its extension; however, as the people themselves are beginning to understand that the enforcement of the necessarily rather annoying rules and restrictions is for their good, and the Government is thoroughly alive to the importance of the matter, we may, I hope, look for better results in the near future.

Etiology and distribution.

The disease has prevailed almost exclusively during the summer and early autumn, although for several recent years it has persisted more or less through October into November, as is emphatically true of the present season.

Low and damp localities are most severely attacked, and rural districts suffer, in proportion to the population, more than do urban.

All ages and both sexes are apparently equally liable to the malady, many cases occurring in young infants.

There is, I think, a period of immunity for those who have experienced an attack, but its length can not yet be determined. A study of the annual statistics of the various provinces throws a little light upon this question of immunity. In 18 of the 23 districts, more or less invaded prior to 1893, I find manifest evidence of a tendency in the disease to recur with severity after a tolerably constant term. Thus, in 12 provinces of the 18, the interval between the dates of greatest prevalence was, distinctly, six years. In 3 this period was seven and in 3 five years. The returns of 3 of these 18 districts showed, in each, 2 instances of a regular interval between the worst outbreaks; in 2 cases of six years each, in the third of seven years each. So far as these data go, they would indicate a period of immunity after attack, varying from five to seven years, the average being six years.

Dysentery does not spread with the rapidity and violence of cholera, but, the germs having reached a given locality, the disease does not cease until the larger proportion of the people there have suffered an attack. As a rule the infection continues very active in the same spot, but from two to four years, a period of comparative immunity following. This latter statement as to a term of exemption is derived from the opinions of the native profession and not from statistical returns.

Susceptibility of the population may be exhausted in the first year's attack, and that locality escape the next season, and, in a province suffering for successive years, the regions of maximum infection vary each year. When the disease returns a second year, in the same place, it appears earlier in the spring than at a primary outbreak, and continues later into the autumn.

The malady is prone to appear first on the slopes of hills or mountains, extending thence to the lower grounds, and from there, perhaps, invading the towns, which are generally situated at a low level.

The epidemic coinciding in its seasonal appearance with the period when certain industries, such as the cultivation of rice and the production of silk, are carried on, the pecuniary loss to the people is, in the case of a severe outbreak, immense. In the small ken of Yamanashi, which suffered very greatly both in 1897 and 1898, the loss to its people in the former year, on silk alone, is estimated at over a million yen, or five hundred thousand dollars, no little sum to a small and poor agricultural population.

The infection of dysentery seems, in some respects, to resemble that of cholera, from which, however, it differs widely in others. Thus it is, unquestionably, often propagated from man to man through the medium either of the evacuations themselves, contaminating food or drink; or, it seems probable, by emanations from the morbid fœces when accumulated in closets or elsewhere; the germs of the malady being, perhaps, capable of transmission through the air to a limited distance.

There are cases in which the infection seems to enter first at the anus, which will be more fully referred to under the symptomatology of the disease. The structure of the Japanese privies, in which the opening for the evacuations is much larger than can be covered by the body, as a result of which there is often a strong upward current of air during the occupation of the closets, would especially favor such a method of infection, particularly if paper is used which has been for a long time in the privy.

Water is, almost certainly, the chief agent in the distribution of the dysenteric poison. This occurs, locally, by the contamination of wells, the principal source of drinking water in Japan, and which are often within a few feet of the closets; or, on a larger scale, by the infection of streams. Japan is an exceedingly well-watered country, thousands of small water courses coming down from the everywhere near mountains toward the sea; and most villages, which generally consist of one long street, have a rivulet running through their length, used, to a greater or less extent, as a domestic supply. Now, night soil being almost the only fertilizer used upon the time-worn lands of the country, and rice, the leading crop, requiring for its culture a constant irrigation of the plots, which often extend in terraces far up the hillsides, each little field receiving in turn the washings of its higher neighbor; the wide distribution of any germs existing in fœces is inevitable. The foregoing, I think, goes far to explain the disproportionate affection of the rural population by dysentery, to which I have already alluded.

It is a curious fact that in the dozen or more epidemics of cholera through which Japan has passed since 1877, although cholera is a water-borne disease, and all the local conditions described apply to it equally with dysentery, the rural population suffered from it in small proportion as compared with the denizens of cities. We have in Japan accounted for this by the assumption that the acid fermentation, to which the night soil is invariably submitted before being placed upon the fields, promptly kills the coma bacillus.

It may be that further investigation will show that the bacillus of dysentery, be it specific or be it a depraved variety of bacterium coli, is more resistent under these circumstances, as well as more capable of enduring winter temperatures, than is that of cholera. This would agree with the observed peculiarities as to the distribution and persistence of the epidemic now under consideration.

As is the case with cholera, plentiful rains increase the violence of the epidemic where it is present, while floods cause its abatement. In the former case the germs from the closets and surrounding soil are washed into the wells or streams, in the latter are swept clean away.

The spreading of infection is favored by conditions, many of which exist, under similar circumstances, everywhere, but are, perhaps, somewhat specially operative in Japan. The people generally are entirely ignorant of the most ordinary hygienic precautions, and rarely, in the rural districts, have any skilled advice available. They use water, which, in the presence of dysenteric disease, is sure to be contaminated, and entertain and receive visits rather more when disease is in the house than at ordinary times. They keenly dread the enforcement of the regulations for the control of the disease, which are necessarily somewhat onerous, not only imposing restrictions on the patient and his family, but seriously interfering with the employment of the latter, upon the daily continuance of which subsistence may depend. Add to this the effect of religious feeling, antagonized by the prohibition of the usual ante and post mortem ceremonies and funeral feast, and inducing, in case of sudden outbreak of disease, the organization of a local religious festival with the idea of placating the supreme powers, which gathers the people from the entire neighborhood, often from localities not yet infected, and is accompanied by feasting, intoxication, and miscellaneous intercourse of sick and well. It is evident that contagion is likely to be widely distributed, and that concealment of the presence of the malady must be very frequent.

Symptoms and course of the disease.

In many instances the distinct manifestation of the malady is preceded by general malaise and anorexia, sometimes accompanied by vomiting, chills, or cramp in the limbs. In other cases the attack begins as a catarrhal diarrhea, which speedily becomes mucous and more or less bloody, the stools at the same time being less copious and more frequent. Even during the catarrhal stage the sigmoid flexture may sometimes be felt to be thickened and hardened. Very many cases are frankly dysenteric from the first, the earlier evacuations mucous and hemorrhagic, and occasionally motions of this kind will, at the very onset of the illness, immediately precede a perfectly normal and healthy stool, suggesting that in these cases the infection has entered by the anus. (See note.)

NOTE.—An epidemic and highly infectious gangrenous disease of the rectum, which may or may not be closely allied to dysentery, has been described by Sigaud (Du Climat et des Malad. du Brezil, Paris, 1844), and Brett (Indian Tribes of Guiana, London, 1868), as occurring in South America; and by Corney, (Trans. Epidemiological Soc. 1887–88), as a serious cause of death in Fiji and other South Sea islands. (See Davidson, Hygiene and Diseases of Warm Climates, Edinburgh, 1893.) This malady, as a rule, begins at the anus itself, which becomes immediately dilated, and progresses rapidly upward until death occurs. I have met with other, and more recent, allusions to this interesting form of rectal disease but am, at present, unable to refer to them. It is of interest in connection with the form of dysentery, and the presumed method of infection, which I have metioned above.

In all cases, as the malady progresses, the stools increase in number though, individually, very small in quantity, until they may pass as often as fifty, sixty, or more times in the twenty-four hours. Hemorrhage is often very profuse even in the first days of sickness, so that the patient becomes very anæmic.

Should the disease continue in severe form, so-called croupous or diphtheritic infiltration of the mucous membrane may occur, followed by ulceration and sloughing, this being rendered externally manifest by the dark color and gangrenous odor of the discharges, which often contain fragments of necrosed mucosa.

Ulcers once established, the stool becomes more or less purulent, a condition that, in chronic cases, may be maintained for many weeks or months.

Pain, in varying degree, is felt in the lower abdomen from the first, in the milder cases temporarily relieved by an evacuation, in the severer forms of the disease almost incessant and excruciating.

Tenesmus is universal and often unbearably severe and constant, continuing, sometimes, after practical recovery.

Tenderness on pressure over the region of the colon exists in all cases, in some limited to the sigmoid tract, in others including the greater part or the whole of the intestine.

As a rule, the earlier manifestation of the malady occurs in the descending colon or upper rectum, and its progress upward can be traced, as the bowel is further involved, until, perhaps, the whole of the large intestine and a portion of the ileum is attacked. Exceptional cases are met with in which the symptoms point distinctly to a descending enteritis, beginning at the coecum and advancing toward the rectum.

The temperature is almost always more or less elevated during the first four or five days of the attack, ranging from 38° C. to 39.5° C., and irregularly fluctuating, with a tendency to an evening rise. In very acute cases the fever may reach 40° C., or even more. After the first five days, however, the temperature often becomes subnormal, from 36° C. to 36.5° C., and continues thus till death, recovery, or the occurrence of some accident in the progress of the case, such as septic irritation which induces a rise in the bodily heat.

The pulse at first varies with the temperature, and later in frequency, volume, and action corresponds to the severity of the special case.

In some instances not very rare the nervous system seems overwhelmed by the direct action of a toxine very early in the attack, and the patients die in collapse, having had comparatively few stools, little or no hemorrhage, and long before their prostrate condition could be brought about by the relatively slow process of exhaustion. I have seen cases at the third to the fifth day of their illness the appearance and general condition of which would immediately suggest cholera to an experienced observer, an impression which would be confirmed by the temperature, generally far below normal, as well as by the small, weak, and frequent pulse, though the specific symptoms and post-mortem appearances clearly proved the disease to be dysenteric. This early intoxication I believe to be rarely or never met with in amœbic dysentery.

Another class of cases which, even more than those just mentioned, deserve the epithet malignant, combine the deep depression of direct intoxication with frequent and exhaustive discharges, active and extensive hemorrhage and early gangrene of the mucous membrane, and speedily prove fatal, delirium coming on before death.

In the greater number of fatal cases death takes place in from seven to ten days. When recovery occurs, the duration of the attack is, usually, from three to four weeks, but should the malady become chronic its continuance is indefinite.

Chronicity is, however, not a very common event of the acute epidemic dysentery of Japan, neither are complications of any kind so frequent that it can be said that the disease specially tends to any such other than those due, perhaps, to contiguity or to accident, such as gastritis or peritonitis.

Abscess of the liver especially, so prone to occur as a sequel of tropical or amœbic dysentery, is so rare, if it ever occurs, after the form of enteritis now under consideration, that I have, after diligent inquiry, been unable to learn of a single undoubted case, and certainly I have never seen one.

From the foregoing it will be seen that the course and symptoms of epidemic dysentery, as existing here, do not essentially differ from those of the same malady elsewhere.

Pathological anatomy.

Necropsis are, in Japan, obtained with great difficulty, and the number of dysenteric cases which have been examined post-mortem is not large. Still, such investigations as have been made, have been conducted by able and qualified men, and the cases indiscriminately selected. The results, probably, afford a fair idea of the average appearances, which, perhaps with the exceptions noted below, are those of acute epidemic dysentery the world over, and which, in a report of this nature, it is scarcely necessary to recapitulate.

The extensively undermined ulcer, so graphically described by Councilman and Lafleur, resulting from extensive destruction of the submucosa is, however, seldom met with, the destruction of the tissues appearing to progress from the surface downward, or to be the effect of general interstitial necrosis from croupous infiltration. As elsewhere, it is the summits of the folds of the large intestine, or those of the valvulæ conniventes of the smaller gut, when this is involved, that are generally earliest attacked. The follicular, pustule-like ulcer is very uncommon. Perforation is unusual, the muscular coat offering a strong resistance to the diseased process.

With regard to the healing of the ulcers, Dr. Shiga, as a result of his observations, is inclined to think that the marked degree of puckering and general contraction of the tissues in cicatrization, perhaps resulting in stricture of the intestines, which is so often met with as a result of dysentery elsewhere, perhaps of that due to amœbæ in particular, is the exception in the disease as at present met with here, even when it has become chronic. Several specimens, which Dr. Shiga was good enough to show me, go far to confirm this conclusion.

The mesenteric glands, corresponding to the affected portion of intestine, are frequently enlarged and hardened and may suppurate; or, if life is sufficiently prolonged, even become caseous. I am under the impression that, in amœbic dysentery, these glands more often escape attack.

Dr. Shiga has found in the mucous membrane about the ulcers the baccillus to which he ascribes the disease.

Bacteriology.

In attempting an intelligent résumé of the more important bacteriological work that has recently been done in connection with epidemic dysentery, I find myself heavily handicapped by the want of any even approximately complete library of reference, being compelled to rely almost entirely upon my own small resources. It is thus very probable that investigations of importance may have escaped my notice. For this, if so, I beg that due allowance may be made.

Disregarding the earlier attempts to detect the material infection of dysentery, the more as in few of these was there any distinct differentiation of the amœbic from other forms, we find that, in 1891, Prof. M. Ogata, of the University of Tokyo, isolated a bacillus from the stools of dysenteric patients which, in culture, produced dysenteriform symptoms and lesions in animals. This bacillus he does not seem to have specifically identified. (Centralblatt für Bacteriologie und Parasitenkunde, 1891.)

Celli and Fioca, and Celli and Scala (Hygien. Institut. Rom. Univ. 1895, and Annal. Exper. Hygiene, n. s., Vol. VI. fasc. 2.), published researches, in their opinion, tending to show that the bacterium coli, perhaps pathogenically modified, is, as indicated by the earlier investigations of Chantemesse and Widal, corrected by Baumgarten and Kartulis among others, the specific cause of dysentery in man. The results of this work of Celli and his colleagues were, later, confirmed by Del Pino and Alessandro. Celli and Fioca have continued their labors, and, in 1897 (Centralblatt für Bacteriologie, 1899), describe the separation of a toxine from cultures of their so-called bacterium coli, var. dysenteriæ, with which they endeavored to immunize dogs and donkeys, succeeding best with the latter, and, after, used also a toxine derived from the same bacillus by their colleague, Valentiti, and which, in carnivora, produced characteristically dysenteric symptoms, again using donkeys as the subjects for immunization. With serum obtained from these animals they treated 7 cases of dysentery, of acute and epidemic nature, with recovery of 6 and death of the 7th, which was complicated by mitral disease and nephritis. Of 4 cases of corresponding severity, treated at the same time by ordinary methods, 3 died.

They tested the agglutinative reaction, upon cultures of their bacillus, of the serum of immunized animals, and of that of a patient just recovered from dysentery, with positive results in both cases. Control experiments with the serum of the healthy were negative. Celli has examined the bacillus dysentericus of Shiga, and compared it with his bacterium coli, var. dysenteriæ, and, apparently, believes the two to be identical, though differing in some reactions. It is for this reason that I have given the foregoing résumé of his work.

Dr. Kiyoshi Shiga, assistant to Professor Kitasato, and in charge of the laboratory of the Tokyo Institute for the Study of Infectious Diseases, has published (in Saikin Gakku Zasshi, Tokyo, December, 1897, and in Centralblatt für Bacteriologie, Band XXIV, Nos. 22, 23, and 24, 1898) the results of his studies of dysentery during the preceding summer and autumn, results which he believes his later investigations, to the present time, have fully confirmed.

Dr. Shiga concludes that he has found the specific cause of dysentery, at least of that now ravaging Japan, in a short rod of about the same size as bacterium coli communis, and, morphologically, very similar to the bacillus of typhoid, having, like the latter, a tendency to produce forms of involution. It is usually solitary but, sometimes, united in pairs, and, with methylene blue, both ends stain thoroughly. After cultivation for some time, however, the staining may be rather irregular. It is easily decolorized by the method of Gram, and does not form spores. It is killed by exposure to a temperature of 68° C. for twenty minutes. The bacilli show no active movements, and no flagella have yet been demonstrated. It does not liquefy gelatin, and grows, at ordinary temperatures, but slowly, but very rapidly at the temperature of the blood. The culture media should be slightly alkaline.

In gelatin plate cultures small, dewdrop-like colonies develop at the end of twenty-four hours, which, under the microscope, are seen to be of regular outline and spherical form, and show, by transmitted light, a granular appearance and a yellowish color. Although these colonies develop day by day, they do not form superficial thin pellicles, like the bacterium coli commune, and surface and deep colonies show no special differences.

In stick culture in gelatin, a greyish white growth of crowded colonies forms along the line of puncture, and no liquefaction occurs.

On nutrient agar-agar, in an incubating oven, large solitary colonies are evident at the end of twenty-four hours, which are bluish white by transmitted light, rounded and moist. If, with a needle, a little material is scraped from their surface, and a stained preparation is made, regularly formed bacilli will be seen. After two days each colony becomes surrounded by a transparent border, which gradually increases in width, and the forms of the bacilli become irregular. Under these conditions they do not stain clearly or uniformly, showing a dotted appearance at their ends.

On glycerin agar-agar the growth is not so favorable as on simple agar-agar. Serum also is not a suitable medium.

Stick culture in grape sugar agar-agar produces a greyish-white growth in line of puncture without development of gas.

On potato, in the incubator, growth is not very evident at the end of twenty-four hours, but, on careful examination, the surface is seen to be dry, white, and lustrous, becoming yellowish-brown in twenty-four hours more, and at the end of a week showing a thick brownish-pink film.

In bouillon the bacillus grows well and the liquid becomes turbid, but no membrane is formed on its surface.

In peptone solution there is no indol reaction.

In litmus lactose the color becomes reddish at the end of twenty-four hours, and remains so permanently. In litmus lactose peptone the red color appears only after the lapse of forty-eight hours.

The bacillus does not coagulate milk or ferment glucose.

With the blood serum of patients suffering from dysentery, or that of those who have recently passed through that disease, agglutinative reaction is well marked; but fails to occur with the serum of the well, those suffering from other maladies, or with the several curative serums, always excepting that of animals immunized to the dysenteric poison, recently produced by the investigator.

Dr. Shiga calls the bacillus above described "Bacillus Dysentericus."

Celli has recently sent to Shiga a culture of his "Bacterum Coli, var. Dysenteriæ," and the latter has examined it and compared it with his own bacillus. He informs me that the culture of Celli differs in its reactions from his, and that the former appears to him to be of bacterium coli commune only, a conclusion in which he is supported by Professor Kitasato.

An important result of the studies of Dr. Shiga, in this direction, is his production of an antitoxic serum for the treatment of dysentery. Using agar-agar cultures twenty-four hours old, dried in vacuo, as the basis of his immunizing fluid, he has experimented, for more than two years, on various herbivora, chiefly the horse and sheep. He finds the latter to become most quickly immune, requiring but one year's treatment, while the horse demands injections for two years' time before ceasing entirely to react to the poison. Once immunized, however, the horse yields a more powerful antitoxin than does the smaller animal. The process of immunization does not, in the herbivora, produce dysenteric or other intestinal symptoms or lesions. The effect of the injections is shown by malaise, loss of appetite, and rise of temperature, at first to 40° C., consequences which, little by little, fail to appear as the treatment is continued.

The serum is prepared, with the usual precautions, from the blood of an immunized animal, and is used by Dr. Shiga, on the human subject, by subcutaneous injection to the amount of from twenty to forty cubic centimeters in twenty-four hours, in divided doses, the quantity given being proportioned to the gravity of the case. As a rule, no injections are required after the second or, occasionally, the third day of treatment, although, in rare cases, the treatment must be longer continued. No manifest reaction takes place at the time of, or soon after the injection, but, in a considerable proportion of cases, transitory urticaria, chiefly in the neighborhood of the punctures, but, sometimes, spreading quite extensively, appears a week later. This is the only unpleasant consequence of the treatment that has been met with, and is, after all, a trifle.

The effect of the serum treatment, more especially upon the number of the stools, is well shown in the accompanying charts taken at random from the records of Dr. Shiga.

Up to November 1, 1899, Dr. Shiga has treated with serum, cases as follows: 1898, in Laboratory Hospital, 65 cases, death rate, 9 per cent; 1899, in Laboratory Hospital, 91 cases, death rate, 8 per cent; 1899, in Hirowo Hospital, 110 cases, death rate, 12 per cent.

During the same period of 1899, there were under ordinary treatment in Tokyo: At Honjo Hospital, 166 cases, death rate, 37.9 per cent; at Hirowo Hospital, 53 cases, death rate, 37.7 per cent; at Komagome Hospital, 398 cases, death rate, 34.7 per cent; in private houses, 1,119 cases, death rate, 28.5 per cent.

The reports of a comparatively limited number of trials of the serum, made elsewhere than in Tokyo, are also most favorable to its efficiency.

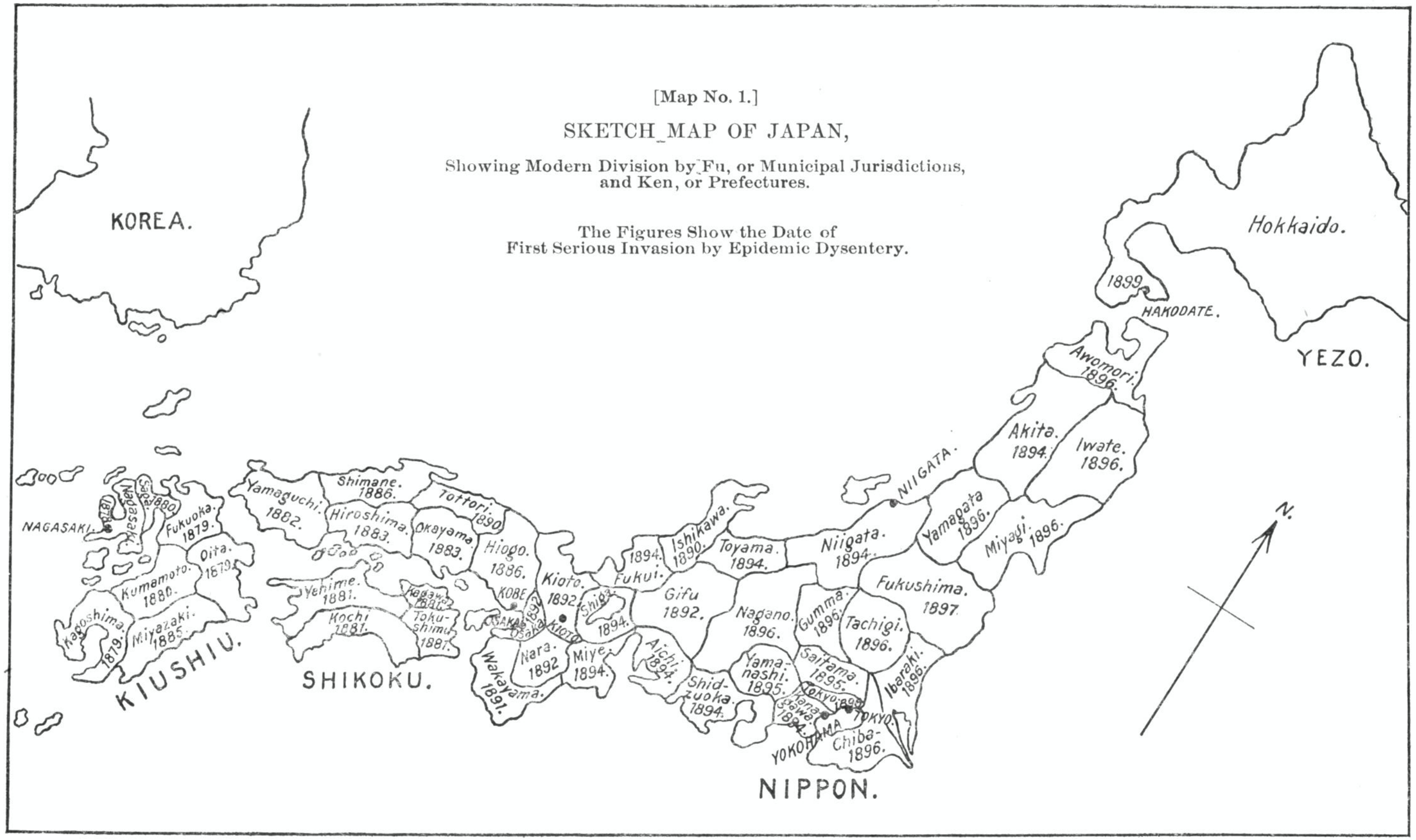
It appears, therefore, that Dr. Shiga's therapeutic work strongly confirms his microscopic and bacteriologic identification of the cause of the existing epidemic.

Whether the serum treatment of dysentery can be rendered sufficiently easy and inexpensive to allow of its very extensive use under existing conditions in Japan, remains to be seen. At present the first cost of the necessary animals and the expense of keeping them during the long term demanded for their immunization, and the comparatively large doses of the serum required, render it impossible that this method of treatment can be largely adopted save by direct and munificent governmental assistance, an attempt to secure which will, undoubtedly, be made before long.

In conclusion, referring to your query as to whether, in my opinion, any of the cases returned as dysentery may be of plague or cholera, I would answer as to plague, emphatically, no. As concerns cholera, it may have been that during seasons when epidemic cholera was present as well as dysentery, which has not been the case since 1894, some slight confusion may have arisen, but only in rare and single cases where, perhaps, the cholera was hemorrhagic in character, or the dysentery of the fulminating and collapsing type to which I have already alluded. Even the rank and file of the Japanese medical profession are thoroughly familiar with the symptoms of the two diseases, by prolonged and repeated experience.

I wish to express my acknowledgments to Dr. Hasegowa Tai, director of the sanitary bureau of the Imperial Government; to Professors Baelz, Ogata, and Kitasato; and to Drs. K. Shiga and K. Rokkaku, for substantial assistance in the preparation of this report.

Respectfully, STUART ELDRIDGE, M. D.,
Acting Assistant Surgeon, U. S. M. H. S.,
Sanitary Inspector, Yokohama.



[Map No. 1.]

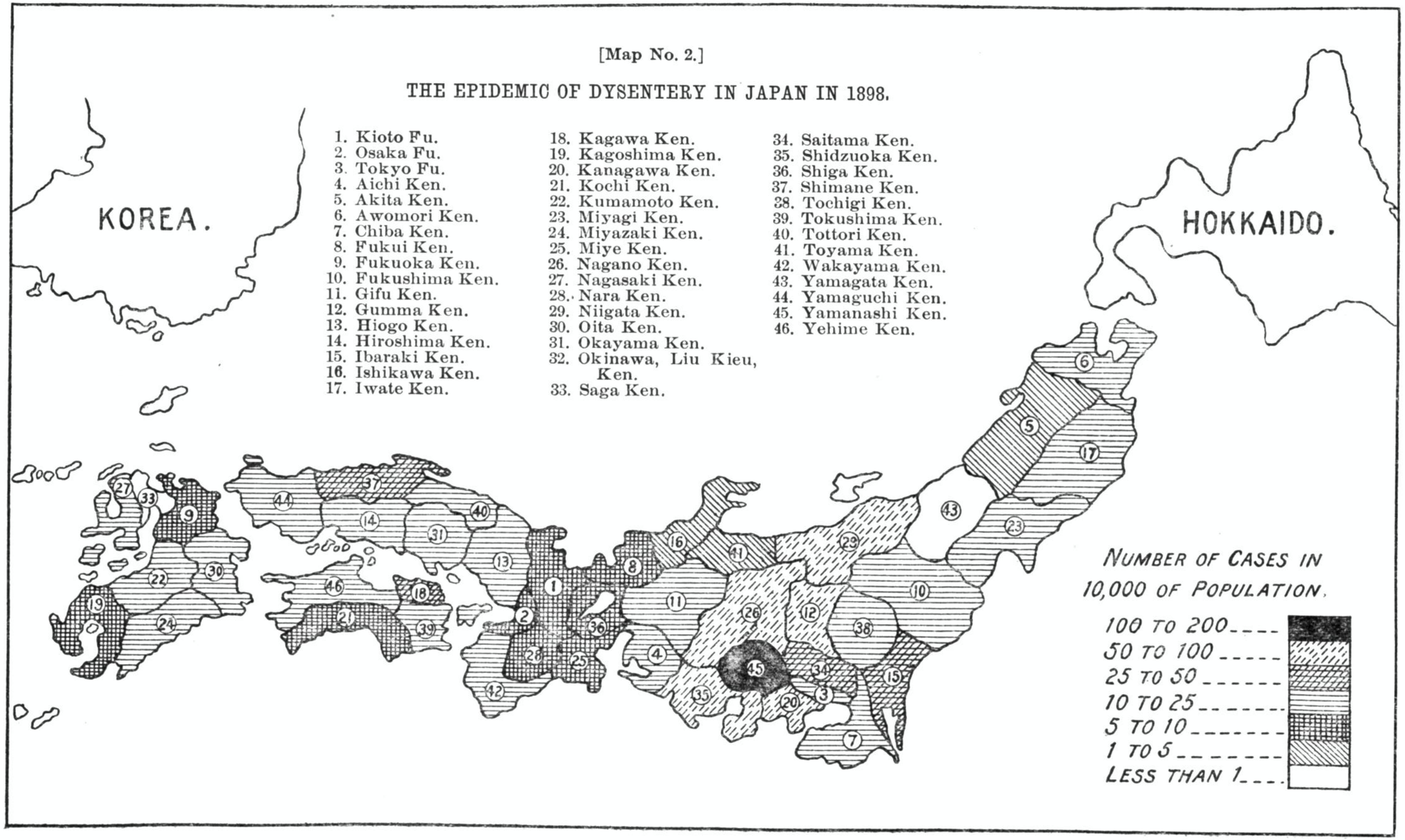
SKETCH MAP OF JAPAN,

Showing Modern Division by Fu, or Municipal Jurisdictions, and Ken, or Prefectures.

The Figures Show the Date of First Serious Invasion by Epidemic Dysentery.

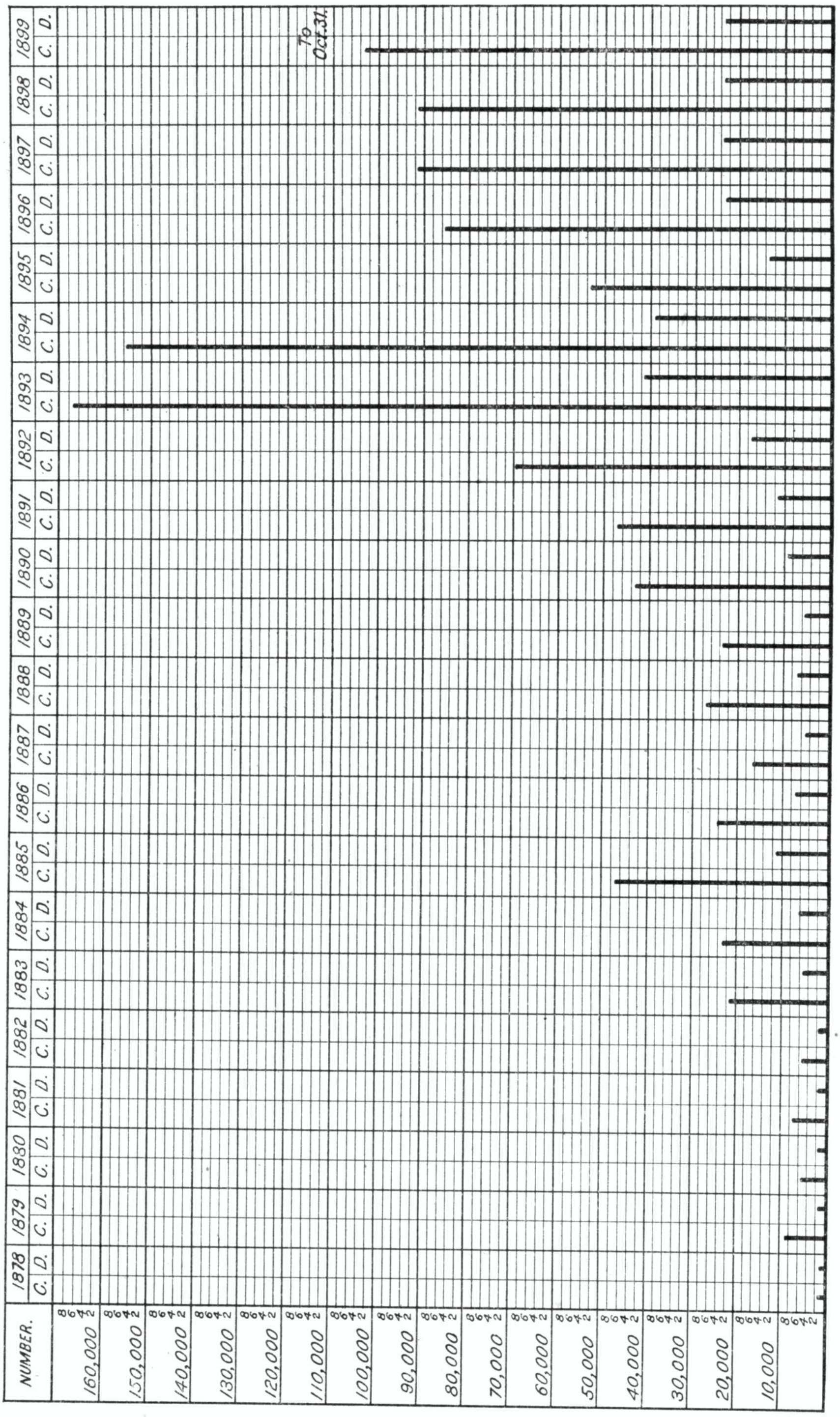
[Map No. 2.]

THE EPIDEMIC OF DYSENTERY IN JAPAN IN 1898.

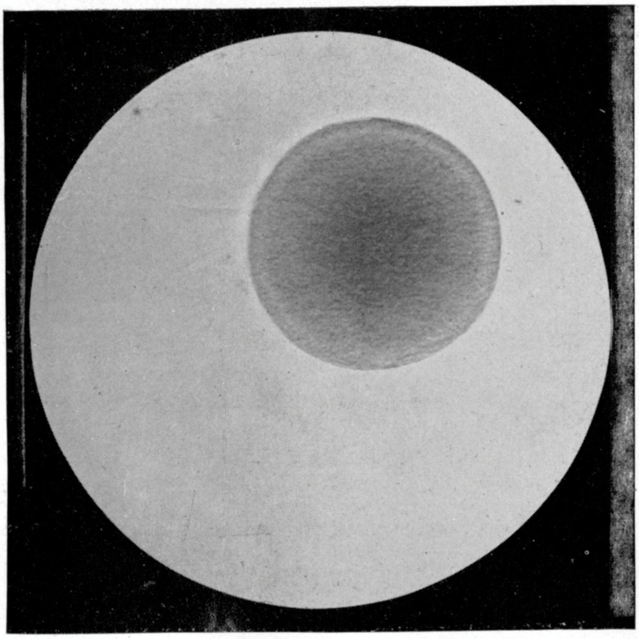


1. Kioto Fu.
2. Osaka Fu.
3. Tokyo Fu.
4. Aichi Ken.
5. Akita Ken.
6. Awomori Ken.
7. Chiba Ken.
8. Fukui Ken.
9. Fukuoka Ken.
10. Fukushima Ken.
11. Gifu Ken.
12. Gumma Ken.
13. Hiogo Ken.
14. Hiroshima Ken.
15. Ibaraki Ken.
16. Ishikawa Ken.
17. Iwate Ken.
18. Kagawa Ken.
19. Kagoshima Ken.
20. Kanagawa Ken.
21. Kochi Ken.
22. Kumamoto Ken.
23. Miyagi Ken.
24. Miyazaki Ken.
25. Miye Ken.
26. Nagano Ken.
27. Nagasaki Ken.
28. Nara Ken.
29. Niigata Ken.
30. Oita Ken.
31. Okayama Ken.
32. Okinawa, Liu Kieu, Ken.
33. Saga Ken.
34. Saitama Ken.
35. Shidzuoka Ken.
36. Shiga Ken.
37. Shimane Ken.
38. Tochigi Ken.
39. Tokushima Ken.
40. Tottori Ken.
41. Toyama Ken.
42. Wakayama Ken.
43. Yamagata Ken.
44. Yamaguchi Ken.
45. Yamanashi Ken.
46. Yehime Ken.

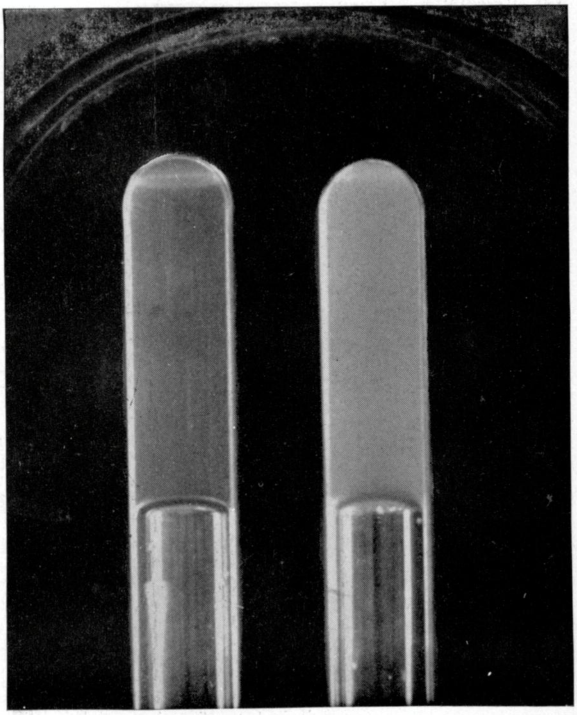
CASES OF AND DEATHS FROM DYSENTERY IN JAPAN, 1878 TO 1899.



BACILLUS DYSENTERICUS.-

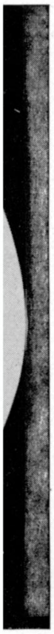


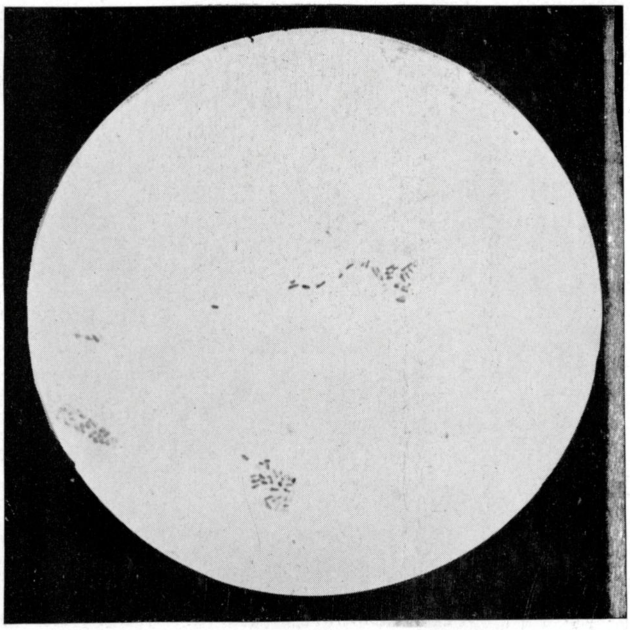
Gelatine Plate Culture, third day.



1. Agglutinative Reaction of Serum of Patient with Dysentery, on Culture in Bouillon.
2. Control Experiment with Serum of Health.

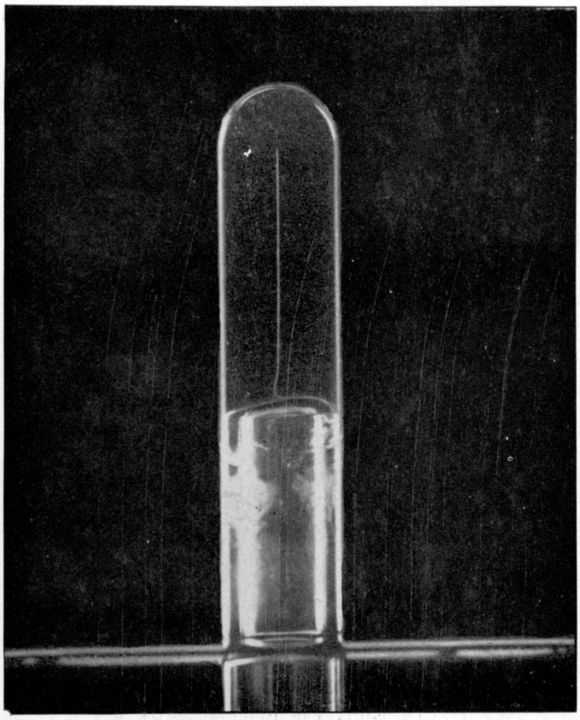
DYSENTERICUS.—SHIGA.





Bacillus Dysentericus.

tery, on



Stick Culture in Gelatine.

TREATMENT OF DYSENTERY BY DR. SHIGA'S SERUM.

Serum treatment of dysentery.—Recovery.